

#### **Research Article**

# New races of *Tilletia laevis* and *Tilletia caries*, the causal agents of wheat common bunt in Khuzestan province, Iran<sup>1</sup>

#### Nahid Albughobeish<sup>1</sup> and S. Ali Moosawi Jorf<sup>2\*</sup>

- 1. Department of Plant Protection, Faculty of Agriculture, Shahid Chamran University, Ahvaz, Iran.
- 2. Department of Plant Pathology, Faculty of Agriculture, Tarbiat Modares University, Tehran, Iran.

**Abstract:** In Iran, common bunt of wheat is one of the most important diseases of wheat and using resistant varieties is the best strategy against it. In order to find resistance sources against the disease for effective breeding programs, it is critical to determine races of the pathogen. In this study, spikes infected with common bunt of wheat were collected from different farms of Khuzestan province in 2005-2006. *Tilletia laevis* and *Tilletia caries* were identified as the causal agents of the disease. Twenty selected isolates were inoculated on differential genotypes and planted under field condition. Fifteen different pathogenic races were identified in this study; L-19, L-21, and L-1 (for *T. laevis*) T-11, T-1, T-2 and T-31 (for *T. caries*). Except L-21 and L-1, other races were reported for the first time in Iran until 2008. Also in this study eight pathogenic races were identified based on virulence/avirulence patterns. Results showed that host resistance genes Bt6 and Bt14 were effective against races of *T. laevis*, and host resistance genes Bt5, Bt6, Bt10 and Bt14 were effective against races of *T. caries* in Khuzestan province.

Keywords: resistance, pathotype, Tilletia foetida, Tilletia tritici, Iran

#### Introduction

The common bunt of wheat has been considered as one of the most significant diseases of wheat which infects all wheat farms. Since the Middle East is deemed as center of wheat, it is also known as the origin of common bunt. As such, after rusts, common bunt is the most widely spread and significant disease in this region (Fisher and Holton, 1957; Wilcoxson and Saari, 1996). The damage resulting from this disease is estimated at 5-7% (Hoffmann, 1982). Even when

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the damage is not significant, infected grains with bunt teliospores are of poor quality and cause problems in wheat trade (Wilcoxson and Saari, 1996). In Iran, the disease is observed in all farms of any district; however, its incidence is high in west and northwest which results in 25-30% damage in infected farms (Mardoukhi and Torabi, 2002).

Although, seed treatment with some fungicides can effectively control soil-borne and seed-borne teliospores, but it is not economical and it also results in environmental pollution. Besides, in semi -dry districts the spores of fungus survive in soil for a long time and can infect wheat seedling in favorable conditions. Thus, complete protection is not possible through disinfested seed in long term. The most important factor in failure of seed treatment is that some physiologic races of the pathogen become resistant to fungicides such as

<sup>\*</sup> Corresponding author, e-mail: moosawiJorf@modares.ac.ir Received: 12 February 2014, Accepted: 13 September 2014 Published online: 4 October 2014

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hexachlorobenzene and tolerant against other fungicides like carboxyl and polychlorobenzene. Hence, the best strategy to control the disease would be to use resistant varieties (Ata-Hossaini *et al.*, 2003).

Up to now, 15 resistant genes (Bt1 to Bt15) in addition to the gene Btz in *Agropyron intermedium* and the gene Btp in wheat variety PI 173438 have been identified and widely used in breeding resistant varieties. Identifying races of the fungus causing the disease in different districts which have virulence factor for one or more of the resistance genes as well as determining effective resistance genes and studying possible virulence changes of the agent fungus is necessary for development of resistant varieties (Mardoukhi and Torabi, 2002).

Race specific pathogenicity of these fungi was first demonstrated in T. caries and T. laevis by Faris (1924). Kendrick (1961), based on of seven standard reactions varieties, categorized 28 races including 10 races of T. laevis and 18 of T. caries into 17 groups; which themselves were grouped based on reactions of one of each six types of resistance genes in varieties Martin, Hussar, Hohenheimer, Ridit and Omar.

Hoffmann and Metzger (1976) identified ten genes against bunt (Bt genes) in standard varieties and called them numbers 1 to 10 (Bt1 to Bt10). Using the proposed formula and the combination of virulence of various isolates of the pathogen, they determined 39 races in northwestern districts of Pacific Ocean. They used monogenic lines as well as some varieties with specific genes such as P.I.178383 with three resistance genes Bt8, Bt9, and Bt10 and P.I.166921 which were resistant against all races in order to study new combination of virulent changes. In their experiments they recognized seven new races of T. caries of which the most widely spread were L-10 and L-16 and then T-1 and T-6.

In Turkey, based on reactions of eight standard varieties, Finci (1981) identified 29 races of *T. laevis* and eight of *T. caries*. In another experiment in 1983 with more samples and using

11 standard varieties, 35 races of *T. laevis* and eight races of *T. caries* were recognized.

Sood and Singh (1985) reported four races of *T. laevis* and two of *T. caries* in India.

In Australia, Andrews (1987) recognized eight races of *T. laevis* and three of *T. caries* based on reactions of 10 standard varieties.

In Syria, Ismail *et al.* (1995) studied 18 spike samples infected with bunt and identified five races T-11, T-13, L-18, L-9, and L-19.

In Iran, the first studies on races of the common bunt of wheat were done by Mardoukhi and Torabi (2002) and four races L-3, L-4, L-10 and L-1 were identified. Ata-Hossaini *et al.* (2003) studied 21 isolates of *T. laevis* from Khorasan province and reported four races L-8, L-3, L-1 and L-20 for the first time in Iran. As well, Daryaie *et al.*, (2007) from Kermanshah province, using nine differential cultivars, identified nine races L-29, L-20, L-10, L-31, L-30, L-35, L-33 and L-32.

Pathogenic races are genetic variants of the same species and can be distinguished by their ability to attack host genotypes with different resistance genes. Thus, the expression of resistance or susceptibility of a wheat cultivar depends on the pathogenic race (s) attacking it. Growing resistant varieties keeps the level of common bunt low, so that other controls are not required. Present study intended to identify races of the causal agents of common bunt of wheat in Khuzestan province.

#### **Materials and Methods**

#### **Collection of common bunt samples**

In order to collect isolates of the causal agents of the fungus, samples were collected from 300 wheat farms in 18 towns of extensively cultivated areas in Khuzestan province in 2005-2006. Any infected spike was considered as an isolate.

#### Pathogen identification

In order to identify species of *T. laevis* and *T. caries*, morphological (macro and microscopic) as well as physiological (teliospore germination) characteristics were studied.

To study the rate of teliospores germination, sori or infected seeds were taken from each isolate (spike) and superficially disinfected by Ethylene 70%. Then teliospores were taken by sterile needle from central part of sorus and uniformly distributed on 1% water Agar medium. The medium was kept in an incubator at  $14 \pm 2$  °C. After seven days, germination reached to its maximum. Germination of 50 spores was studied in four locations of each petri dish.

#### **Selection of isolates**

Because it was not possible to study all collected isolates from different regions of Khuzestan province, therefore, after determining species, only the isolates with highest percent germination were selected for race identification.

#### **Differential genotypes**

Differential wheat genotypes including 15 spring wheat cultivars harboring resistant genes Bt0, Bt1, Bt2, Bt3, Bt4, Bt5, Bt5, Bt6, Bt7, Bt8, Bt8, Bt9, Bt10, Bt14 and Bt15 (Table 2) were prepared from Seed and Plant Improvement Institute of Karaj, Iran, and used for this study. Spring line series are mostly made by combination of resistant cultivars with line Red Bobe which is susceptible to bunt. Among these cultivars, there are two durum wheat named Doubbi with gene Bt14 and Carlton with gene Bt15 (Mardoukhi and Torabi, 2002). In spring line series, Red Bobs with nonresistant gene (Bt0) is used as international control.

#### **Inoculum preparation**

An infected spike from each sample was selected as a fungus isolate for determining races and virulence factors. In order to make fungus inoculums, infected seeds were grinded in a Chinese mortar and screened through a 35 µm mesh sieve to remove remnants and to obtain pure teliospores of each isolate.

#### **Inoculation of differential genotypes**

Seven Grams of seeds of differential cultivars for each combination of cultivar-isolate was first disinfested with sodium hypochlorite 5% for 5 min. Then, they were rinsed and dried under sterile hood. Afterward, each differential genotype was

mixed with teliospores of each isolate by the weight ratio of 5 in 1000 (5 grams of spores in 1000 grams of seeds) in separate envelopes. The envelopes were strongly shaken for 5 min so that the spores equally covered the seeds. In this way, any 1 g seed was mixed with about 80000 teliospores (Andrew, 1987; Siddeque Mirza and Arshadkhan, 1983). In order to preserve viability of teliospores germination, inoculum preparations and seed inoculations were done a day before planting.

#### Planting and identification of pathogenic races

Any group of differential genotype inoculated with a specific isolate was planted separately in a field nursery. Each inoculated cultivar was sown by hand at a depth of 4cm in two 50 cm rows. Planting was done during mid-December of 2006 in experimental farm of Agricultural College, Shahid Chamran University, Ahvaz, Iran. Initial irrigation and the farming practices were done according to norm of the area. Then all harvestable spikes of each cultivar-isolate interactions were carried to the lab and percent of infected spikes were determined. Regarding reaction of differential genotype to isolates, 10 percent infections or less were defined as avirulent-resistant and more than 10 percent were considered virulent-susceptible for the related resistance gene in that wheat genotype (Hoffmann and Metzger, 1976).

#### **Results**

## Selection of isolates for identification of pathogenic races

Among 300 collected isolates from wheat fields of Khuzestan province, 200 isolates were identified as T. laevis and 100 isolates as T. caries, based on teliospore morphology and germinability of teliospores on 1% water agar at  $14 \pm 2$  °C within 7 days (Fig. 1). Twenty isolates with better germination vigor (50 to 80%) were selected for race identification (Table 1).

### **Evaluation of differential genotypes and identification of pathogenic races**

Differences in the reaction of differential cultivars to different selected isolates were observed Table 2).

The races of the 20 isolates of *T. laevis* and *T. caries* used in this study, were determined by analyzing their virulence/avirulence pattern to a set of differential spring cultivars and based on identification code of physiologic races of the two smut fungi by Hoffmann and Metzger (1976) as well as Ismail *et al.* (1995) (Table 3).

All of the races identified during the survey were virulent on the susceptible cultivar Bt0.

**Isolates 2 and 3:** These isolates which were collected from Rostam Abad village of BaghMalek caused infection in 50% of susceptible cultivar Bt0. They were virulent on cultivar with resistance genes Bt3, Bt2 and Bt4 and avirulent on cultivars having resistance genes Bt1, 5, 6, 7, 8, 9, 10, 14, and 15. According to virulence/avirulence patterns, these isolates were identified as L-19 race (table3).

**Isolate 4:** collected from Rostam Abad village of BaghMalek infected 60% of susceptible cultivar Bt0 and was virulent on cultivars with resistance genes Bt1, Bt2, Bt3, Bt5, Bt8, Bt9 and Bt15 and avirulent on cultivars having resistance genes Bt4, 6, 7, 10, and 14. This unique virulence/avirulence pattern indicates a new race of this isolate of the fungus.

**Isolate 1 and 5:** collected from Mish Darre Zir village of Lali infected 50% of susceptible cultivar Bt0 and were virulent on cultivars with resistance genes Bt1, Bt2, Bt3, Bt4, Bt5, Bt7 and Bt15 and avirulent on cultivars having resistance genes Bt6, 8, 9, 10, and 14. This unique virulence pattern indicates a new race of this fungus which has not been reported yet.

**Isolate 6:** collected from Rostam Abad village of BaghMalek infected 46% of genotype Bt0 and was virulent on cultivars with resistance genes Bt2 This unique virulence/avirulence pattern (Bt0,1, 4, 5, 6, 7, 8, 9, 10, 14, 15) indicates a new race for Iran and was identified as T-11.

**Isolate 7:** The isolate which was collected from village Darre Buri of Lali from cultivar Shata infected 36% of cultivar Bt0 and was virulent only on cultivars with resistance gene Bt7. Its race was identified as T-1.

**Isolates 8 and 10:** collected from village Darre Buri of Lali from cultivar Shata infected 35% of genotype Bt0 and were only virulent on

cultivars with resistance genes Bt1, Bt7 and Bt15 and therefore were identified as raceT-2.

**Isolates 9 and 11:** collected from Darre Buri village of Lali from cultivar Shata infected 30% of the susceptible cultivar Bt0 and were only virulent on the cultivar with resistant gene Bt15. This race with such virulence pattern has not been named.

**Isolate 12:** infected 50% of susceptible cultivar Bt0 and was only virulent on cultivar with resistant gene Bt3. This unique virulence/avirulence pattern indicates a new race. **Isolate 13 and 14:** collected from Darre Buri village of Lali infected 34% of cultivar Bt0 and were only virulent on cultivars with resistant genes Bt2, Bt3 and Bt4. Accordingly their race was identified as T-13.

**Isolate 15:** collected from Darre Buri village, infected 34% of cultivar Bt0 and was virulent on cultivars with resistance genes Bt2, Bt4, Bt7, Bt8 and Bt9 and avirulent on cultivar having resistance genes Bt1, 3, 5, 6, 10, 14, and 15. This unique virulence/avirulence pattern indicates a new race.

**Isolate 16:** infected 44% of susceptible cultivar Bt0 and was virulent on cultivar with resistant gene Bt4. This unique virulence pattern indicates a new race.

**Isolate 17:** infected 65% of cultivar Bt0 and was virulent on cultivars with resistant genes Bt2, Bt3, Bt7, Bt8 and Bt9 and avirulent on cultivar having resistance genes Bt1, 4, 5, 6, 10, 14, and 15. Its race was identified as L-21.

**Isolate 18:** infected 30% of susceptible cultivar Bt0 and was virulent on cultivars with resistance genes Bt1, Bt2, Bt3 and Bt4 and avirulent on cultivar having resistance genes Bt5, 6, 7, 8, 9, 10, 14, and 15. This unique virulence pattern indicates a new race.

**Isolate 19:** infected 40% of susceptible cultivar Bt0 and was 25% virulent only on cultivar with resistant gene Bt7. and was identified as race L-1.

**Isolate 20:** infected 33% of susceptible cultivar Bt0 and was virulent on cultivars with resistance genes Bt3, Bt5, Bt8 and Bt9 and avirulent on cultivars having resistance genes Bt1, 2, 4, 6, 7, 10, 14, and 15. This unique virulence/avirulence pattern indicates a new race of this fungus.

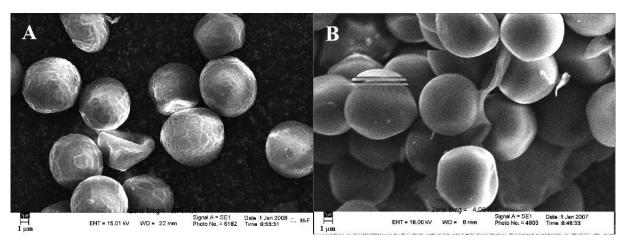


Figure 1 Teliospore Characteristics of *Tilletia caries* (A) and *Tm laevis* (B) by SEM Microscopy (original pictures).

Table 1 Characteristics of selected isolates used for race identification of wheat common bunt.

Isolate No.	Place of sample collection	Date of Sampling	Host cultivar	Species	Germination (%)
1	Lali/Mish dare zir	May 2005	Chenab	T. laevis	80.33
2	Bagh malek/Rostam bad	May 2005	Chenab	T. laevis	81.00
3	Bagh malek/Rostam bad	May 2005	Chenab	T. laevis	75.33
4	Bagh malek/Rostam bad	May 2005	Chenab	T. laevis	81.00
5	Lali/Mish dare zir	May 2005	Chenab	T. laevis	79.33
6	Bagh malek/Rostam bad	May 2005	Chenab	T. caries	64.83
7	Lali/Dare Boori	May 2006	Shata	T. caries	59.16
8	Lali/Dare Boori	May 2006	Shata	T. caries	50.00
9	Lali/Dare Boori	May 2006	Shata	T. caries	62.66
10	Lali/Dare Boori	May 2006	Shata	T. caries	54.33
11	Lali/Dare Boori	May 2006	Shata	T. caries	70.00
12	Lali/Dare Boori	May 2006	Chenab	T. caries	68.33
13	Lali/Dare Boori	May 2006	Chenab	T. caries	66.33
14	Lali/Dare Boori	May 2006	Chenab	T. caries	61.00
15	Lali/Dare Boori	May 2006	Chenab	T. caries	51.00
16	Lali/Dare Boori	May 2006	Chenab	T. caries	79.33
17	Andimeshk/Shaveer	May 2006	Zagros	T. laevis	75.66
18	Andimeshk/Shaveer	May 2006	Zagros	T. laevis	50.00
19	Lali/Dare Boori	May 2006	Chenab	T. laevis	80.33
20	Lali/Dare Boori	May 2006	Chenab	T. laevis	81.66

Table 2 Interactions of differential cultivars with different isolates of wheat common bunt.

Differential cultivar (Spring type)	Isolate No.																			
(1 0 31 )	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Red bobs (Bt0)	V	V	V	V	V	V	V	V	V	V	V	V	V	V	V	V	V	V	V	V
RB-WF 38 (Bt1)	V	A	A	V	V	A	A	V	A	V	A	A	A	A	A	A	A	V	A	A
RB/SEL 1403 (Bt2)	V	V	V	V	V	V	A	A	A	A	A	A	V	V	V	A	V	V	A	A
RB/RDT (Bt3)	V	V	V	V	V	V	A	A	A	A	A	V	V	V	A	A	V	V	A	V
RB/TK3055 (Bt4)	V	V	V	A	V	A	A	A	A	A	A	A	V	V	V	V	A	V	A	A
M82-34 (Bt5)	V	A	A	V	V	A	A	A	A	A	A	A	A	A	A	A	A	A	A	V
RB / Hohenheimer (Bt	5)V	A	A	V	V	A	A	A	A	A	A	A	A	A	A	A	A	A	A	V
RB/RDT (Bt6)	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A
RB/TK3055 (Bt7)	V	A	A	A	V	A	V	V	A	V	A	A	A	A	V	A	V	A	V	A
RB/PI178210 (Bt8)	A	A	A	V	A	A	A	A	A	A	A	A	A	A	V	A	V	A	A	V
RB/PI178210 (Bt8)	A	A	A	V	A	A	A	A	A	A	A	A	A	A	V	A	V	A	A	V
RB/C77090 (Bt9)	A	A	A	A	V	A	A	A	A	A	A	A	A	A	V	A	V	A	A	V
SEL.M83-162 (Bt10)	Α	Α	A	A	Α	A	Α	Α	A	A	A	A	A	A	A	A	A	A	A	A
Doubbi, DW (Bt14)	A	Α	Α	Α	Α	Α	Α	Α	Α	A	A	A	A	A	A	A	A	A	A	A
Carleton,DW (Bt15)	V	A	A	V	V	A	A	V	V	V	V	A	A	A	A	A	A	A	A	A

Table 3 Virulence/avirulence pattern of wheat common bunt races toward host resistance genes (Bt).

Species	Isolate No.	Virulence/Avirulence	Races
Tilletia laevis	2,3	Bt0,2,3,4/1,5,6,7,8,9,10,14,15	L-19
Tilletia laevis	1,5	Bt0,1,2,3,4,5,7,15/6,8,9,10,14	-
Tilletia laevis	4	Bt0,1,2,3,5,8,9,15/4,6,7,10,14	-
Tilletia laevis	17	Bt0,2,3,7,8,9/1,4,5,6,10,14,15	L-21
Tilletia laevis	18	Bt0,1,2,3,4/5,6,7,8,9,10,14,15	
Tilletia laevis	19	Bt0,7/1,2,3,4,5,6, 8,9,10,14,15	L-1
Tilletia laevis	20	Bt0,3,5,8,9/1,2,4,6,7,10,14,15	-
Tilletia caries	6	Bt0,2,3/1,4,5,6,7,8,9,10,14,15	T-11
Tilletia caries	7	Bt0,7/1,2,3,,4,5,6,8,9,10,14,15	T-1
Tilletia caries	8,10	Bt0,1,7,15/2,3,4,5,6,8,9,10,14	T-2
Tilletia caries	13,14	Bt0,2,3,4/1,5,6,7,8,9,10,14,15	T-31
Tilletia caries	15	Bt0,2,4,7,8,9/1,3,5,6,10,14,15	-
Tilletia caries	16	Bt0,4,/Bt1,2,3,5,6,7,8,9,10,14,15	-
Tilletia caries	12	Bt0,3,/Bt1,2,4,5,6,7,8,9,10,14,15	-
Tilletia caries	9,11	Bt0,15/Bt1,2,3,4,5,6,7,8,9,10,14	-

#### **Discussion**

Application of Fungicides, seed disinfestation and cultural practices are effective control measures for smut control, but they are present expensive and may problems with associated toxicity, environmental hazards, and availability or distribution. Furthermore, chemicals may not control the disease as effectively as use of resistant cultivars. Besides continuous exposure to fungicides may culminate in appearance of resistant races of fungus to fungicides. So the development of bunt-resistant cultivars may be the best method to control the disease when resistance sources are available.

It seems that identifying resistance sources and planting resistant varieties are the most appropriate and trustful way to control the disease. Resistance may be overcome by the selective increase of virulent races or by the development of new combinations of virulence genes in the bunt population (Kendrick, 1961).

Evaluation of cultivar resistance is possible artificially infecting them with live teliospores of the causal agents and planting in resistance exploration nursery. resistant varieties without determining races or virulence factors of the pathogen is a shot in the dark, and makes using the varieties limited and vague; because a resistant variety against special races in a determined region may not necessarily be resistant in another region or against other races. Therefore, in order to accurately study resistance of varieties and to produce varieties with resistance in different regions, it is needed to obtain necessary information about pathogenic races and virulence factors of the pathogen, and resistant genes of the varieties.

Identification of resistance genes in host genotype and pathogenic races in the pathogen is based on gene for gene interaction that exists between specific avirulence genes in the pathogen and bunt resistance (Bt) genes in wheat, each race has its own unique virulence pattern. Infection pattern of winter wheat by *T. controversa* (Vahabzadeh *et. al.*, 2004), the

dwarf bunt pathogen is very closely related to *T. caries* and *T. laevis* and that shares a gene for gene interaction with the same common bunt Bt–genes. Having information about genetic resistance of varieties and genetics of pathogenic races, we can recommend varieties with effective resistance genes for each region.

In this study 15 races were identified. Except L-21 and L-1, all of the other races were reported from Iran for the first time until 2008. L-19 collected from Rostam Abad of BaghMalek was virulent on resistance genes Bt2, Bt3 and Bt4. Frequency of this race among identified isolates was 10%. It was for the first time identified by Ismail *et al.* (1995). It was reported in this study for the first time in Iran.

L-21 found in Shavour of Andimeshk with 5% frequency was virulent on resistance genes Bt2, Bt3, Bt7, Bt8 and Bt9. It was for the first time identified as a new race for Iran by Mardoukhi and Torabi (2003).

L-1 with 5% frequency was found in Darre Buri of Lali. It was only virulent on resistance genes Bt7. It was reported by Hoffmann and Metzger (1976) from United States, Andrew (1987) from Australia, Finci (1981) from Turkey, Mardoukhi and Torabi (2002) from west and northwest parts of Iran and Karaj, and Ata-Hossaini *et al.*, (2003) from Khorasan, Iran.

T-11 was found in Baghmalek with 5% frequency, was virulent on resistance genes Bt2 and Bt3. This race was identified by Hoffmann and Metzger (1976) in the United States and Ismail, *et al.* (1995) in Syria.

T-1 found in BaghMalek with 5% frequency was only virulent on resistance genes Bt7. It was identified by Hoffmann and Metzger (1976) in the United States. It was reported that L-16, L-10, T-1, and T-6 were most widespread races in West Canada (Gaudet and Puchalski, 1989).

T-2 found in village Darre Buri of Lali with 10% frequency, was virulent on resistance genes Bt1, Bt7 and Bt15. It was firstly identified by Hoffmann and Metzger (1976).

T-31 found in village Darre Buri of Lali with 10% frequency. It was virulent on

resistance genes Bt2, Bt3 and Bt4. It was for the first time identified by Ismail *et al.* (1995).

Eight other races were identified in the present study based on their unique virulence/avirulence pattern.

Races in village Rostam Abad in BaghMalek found in this study, were virulent on susceptible genes Bt2, Bt15, Bt9, Bt8, Bt5, Bt4, Bt3, and Bt1 with frequencies of %100, %25, %25, %25, %25, %50, %100 and 20% respectively. Therefore, effective resistance genes are Bt7, Bt6, Bt10 and Bt14 that may be used for development of bunt-resistant cultivars in this region.

Races found in Andimeshk were virulent on susceptible genes Bt3, Bt2, Bt9, Bt1, Bt8, Bt7, Bt4, and Bt4 with frequencies of %100, %100, %50, %50, %50, %50 and 50% respectively. So, effective resistance genes are Bt5, Bt15, Bt6 &Bt10 and Bt14 that may be used for development of bunt-resistant cultivars in this region.

The races found in the villages Darre Buri and Mish Darre of Lali, were virulent on susceptible genes Bt7, Bt3, Bt4, Bt15, Bt1, Bt2, Bt8, Bt9 and Bt5 with frequencies of %43, %36, %36, %36, %36, %36, %29, %29, %14 and 14% respectively. Therefore effective resistance genes that might be used for development of bunt-resistant cultivars in this region would be Bt10, Bt6 and Bt14.

Investigations in Iraq have shown that effective resistance genes are Bt4–6, 8–12, 14 (Al-Maaroof *et al.*, 2006).

Lately in a study on identification of pathogenic races of wheat common bunt using differential lines in Lorestan province of Iran the races of L19, T1 and T2 were identified (Noruzi, *et. al.*, 2012),.

In a more recent survey Elyasi, and Farrokhi-Nejad, (2013) studied virulence of *Tilletia laevis* in Khuzestan and Lorestan provinces of Iran. In their study, they identified 20 races of *T. laevis*, the most prevalent of which were L-32 and L-35. Genes Bt7, Bt1, Bt2, Bt3, Bt4 and Bt6 were ineffective against most of the races identified from these two provinces. Most of

the races cannot infect cultivars carrying genes Bt5, Bt8, Bt9, Bt13, Bt14 and Bt15, However, Genes Bt10, Bt11 and Bt12 were effective against all races.

In this study, it was found that host resistance genes Bt6 and Bt14 were effective against races of *T. laevis*, while host resistance genes Bt5, Bt6, Bt10 and Bt14 were effective against races of *T. caries* in Khuzestan province.

Results showed that race variations in common bunt are very great in Iran. Therefore, it is necessary to identify and monitor the pathogenic races of the local populations. Studies on identifying races and virulence factors of the pathogen should be continuous so that when new races are identified, suitable resistant gene sources can be used.

#### References

Al-Maaroof, E. M., Shams-Allah, S. A. and Hassan, M. S. 2006. Current status of wheat bunt disease in Iraq. Czech Journal of Genetics and Plant Breeding, 42: 45-50.

Andrews, J. A. 1987. The bread wheat races of bunt represented in Australian bunt collection. Euphytica, 36: 557-580.

Ata-Hossaini, S. M., Torabi, M. and Jafarpour, B. 2003. Physiologic races of *Tilletia laevis* in Khorasan. Seed and Plant, 18(4):383-393.

Daryaei, A., Ghazali Biglar, H., Haghparast, R. and Rajabi, R. 2007. Evaluation of advanced bread wheat genotypes for resistant to common bunt (*Tilletia laevis* Kuhn) in KermanShah province. Agricultural Sciences and Technology, 21(2):37-46.

Elyasi-Gomari1, S. and Farrokhi-Nejad, R. 2013. Virulence of *Tilletia laevis* in two most important provinces of Iran. International Journal of Agronomy and Plant Production, 4(11): 2953-2959.

Faris, J. A. 1924. Factor influencing the infection of wheat by *T. laevis* and *T. tiritici*. Mycologia, 16: 259-282.

Finci, S. (1981). Studies on the Pathogenic races of *Tilletia foetida* and *T. caries* and their

- pathogenicity on some wheat varieties in Turkey. Eppo Bulletin, 11: 77-82.
- Fisher, G. W. and Holton, C. S. 1957. Biology and Control of Smut Fungi. Ronald Press CO. University of Wisconsin, Madison.
- Gaudet, D. A. and Puchalski, B. J. 1989. Status of bunt resistance in western Canadian spring wheat and triticale. Canadian Journal Plant Science, 69: 797-804.
- Hoffmann, J. A. 1982. Bunt of wheat. Plant Disease, 66: 979-986.
- Hoffmann, J. A. and Metzger, R. J. 1976. Current status of virulence genes and pathogenic races of the wheat bunt fungi in north western USA. Phytopathology, 66: 657-660.
- Ismail, S. F., Mamluk, O. F. and Azmeh, M. F. 1995. New pathotypes of common bunt of wheat from Syria. Phytopathologia Mediterranea 34: 1-6.
- Kendrick, E. L. 1961. Race groups of *Tilletia* caries and *Tilletia foetida* for varietial resistance testing. Phytopathology, 51: 537-540.
- Mardoukhi, V. and Torabi, M. 2002. Identification of pathogenic races of *Tilletia laevis*, the causal agent of wheat common

- bunt in different parts of Iran. Seed and Plant, 18 (3): 362-378.
- Mardoukhi, V. and Torabi, M. 2003. New pathotypes of *Tilletia laevis* from Iran. Seed and Plant, 19 (2): 2-2.
- Noruzi, Z., Mahinpoo, V., Fayazi, F., Mohamedi, M., Jalali, F., Sohilinejad, A. and Darvishnia, M. 2012. Identification of pathogenic races of wheat common bunt using differential lines in Lorestan province. Agricultural Science and Technology, 4(2): 154-158.
- Siddique Mirza, M. and Arshad Khan, M. 1983. A new race L-2 of *Tilletia foetida* from Pakistan. Agricultural Research, 4: 37-40.
- Sood, A. K. and Singh, B. M. 1985. Occurrence and distribution of virulences of *Tilletia foetida* and *T.caries* causing bunt of wheat in Himachol Pradesh. Indian phytopathology, 38: 695-699.
- Vahabzadeh M., Saci-Ahan J. and Sedighi S. 2004. Wheat Field Guidebook. Ministry of Jehad-Keshavarzi, Tehran.
- Wilcoxon, R. D. and Saari, E. E. 1996. Bunt and Smut Diseases of Wheat, Concepts and Methods of Disease Management. CIMMYT, Mexico. D. F., Mexico.

### نژادهای جدید Tilletia laevis و Tilletia caries عوامل سیاهک پنهان معمولی گندم در استان خوزستان

#### ناهید آلبوغبیش و سیدعلی موسوی جرف \*\*

۱- دانشگاه شهید چمران اهواز، اهواز، ایران.

۲- گروه بیماریشناسی گیاهی، دانشکده کشاورزی، دانشگاه تربیت مدرس، تهران، ایران.

\* پست الکترونیکی نویسنده مسئول مکاتبه: moosawiJorf@modares.ac.ir

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